Cardiovascular responses to apneic facial immersion during altered cardiac filling

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Journeay, W. Shane, Francis D. Reardon, and Glen P. Kenny. Cardiovascular responses to apneic facial immersion during altered cardiac filling. J Appl Physiol 94: 2249–2254, 2003. First published February 21, 2003; 10.1152/japplphysiol.01140.2002.—The hypothesis that reduced cardiac filling, as a result of lower body negative pressure (LBNP) and postexercise hypotension (PEH), would attenuate the reflex changes to heart rate (HR), skin blood flow (SkBF), and mean arterial pressure (MAP) normally induced by facial immersion was tested. The purpose of this study was to investigate the cardiovascular control mechanisms associated with apneic facial immersion during different cardiovascular challenges. Six subjects randomly performed 30-s apneic facial immersions in 6.0 ± 1.2°C water under the following conditions: 1) −20 mmHg LBNP, 2) +40 mmHg lower body positive pressure (LBPP), 3) during a period of PEH, and 4) normal resting (control). Measurements included SkBF at one acral (distal phalanx of the thumb) and one nonacral region of skin (ventral forearm), HR, and MAP. Facial immersion reduced HR and SkBF at both sites and increased MAP under all conditions (P < 0.05). Reduced cardiac filling during LBNP and PEH significantly attenuated the absolute HR nadir observed during the control immersion (P < 0.05). The LBPP condition did not result in a lower HR nadir than control but did result in a nadir significantly lower than that of the LBNP and PEH conditions (P < 0.05). No differences were observed in either SkBF or MAP between conditions; however, the magnitude of SkBF reduction was greater at the acral site than at the nonacral site for all conditions (P < 0.05). These results suggest that the cardiac parasympathetic response during facial immersion can be attenuated when cardiac filling is compromised.

diving response; skin blood flow; lower body pressure; blood pressure

THE HUMAN DIVING RESPONSE is initiated by apnea and is augmented by facial immersion in cold water (14). This reflex response is characterized by a reduction in heart rate (HR) through vagus nerve action and by α-adrenergic vasoconstriction in selected vascular beds (2, 6, 14). Superficial cold receptors that are innervated by the ophthalmic branch of the trigeminal nerve enhance the cardiovagal activity involved in the response (18, 22, 31).

Although the mechanism of diving bradycardia is known to be parasympathetically mediated, it is unclear at this time how the response is changed during altered cardiac filling. Only a few studies have implicated reduced cardiac filling in the attenuation of bradycardia. Arnold and Nadel (5) demonstrated that diving bradycardia can be attenuated subsequent to an increased thermal load induced by exogenous heating in 68°C ambient heat. They contended that the attenuation of diving bradycardia occurs as a result of reduced cardiac filling due to peripheral redistribution of blood volume. It has also been demonstrated that high intrathoracic pressure may play a role in the degree of bradycardia observed. Andersson et al. (1) observed that the bradycardic effect was attenuated during periods of high intrathoracic pressure created at 85–100% of prone vital capacity. They concluded that high intrathoracic pressure occluded venous return impeding cardiac filling and thus attenuated the development of diving bradycardia. Although it has been suggested that adequate cardiac filling is a requirement for the development of the diving response, it has yet to be demonstrated directly. Because there appears to be a relationship between cardiac filling and the magnitude of diving bradycardia observed, manipulating factors that alter cardiac filling (postexercise hypotension (PEH), lower body pressure (LBPP)) may alter the bradycardic response.

Cardiac filling can be altered via different techniques. Application of mild LBNP reduces cardiac filling pressure through venous pooling (30, 33), unloads cardiopulmonary baroreceptors (19, 20), and increases efferent sympathetic activity (7). During PEH, mean arterial pressure (MAP) is reduced via both neural and vascular phenomena (15–17). PEH is thought to occur as result of venous blood pooling in the previously active musculature, and its magnitude varies directly with the postexercise elevation in esophageal temperature (TEso) (8, 23). In the PEH period, removal of the muscle pump and the seated posture has transient deficits of return over output as blood accumulates in the venous capacitance system, thus compromising preload (15, 24). In contrast, lower body positive pressure (LBPP) creates a greater venous pressure gradi-
ent that tends to increase cardiac filling, load cardio-
diagnostic activity (13).

Thus the purpose of this study was to investigate the
fluence of cardiac filling on the diving reflex. Specif-
ically, the study was undertaken to examine the inter-
action of the parasympathetic response arising from
face immersion and the simultaneous challenge cre-
ated by the three experimental conditions of modified
cardiac filling. It was hypothesized that reduced car-
diac filling as a result of LBNP and PEH would attenu-
ate the reflex changes to HR, skin blood flow (SkBF),
and MAP normally induced by facial immersion.

METHODS

Subjects

Six healthy, physically active men volunteered and gave
written consent to participate in this study. The study was
approved by the Research Ethics Board of the University of
Ottawa. Five to seven days before the experiments, maximal
oxygen consumption ($\dot{V}O_2\text{max}$) was measured during a pro-
gressive treadmill protocol. The $\dot{V}O_2\text{max}$ data were used to
select the submaximal workload for the experimental exer-

Experimental Protocol

Each subject performed a total of four experimental trials
carried out in random order. The first experiment was con-
ducted after a 36-h period during which subjects were in-
structed to avoid physical activity and excessive stressors
such as exposure to hot or cold temperatures, particularly
during the period between awakening and experimentation
and during transit from home to the laboratory. Further-
more, they were asked to fast at least 4 h before experimen-
tation, and water ingestion was permitted during this time.

Subjects were required to come to the laboratory for two
experimental sessions at an interval of not less than 48 h.
During the first experimental session, the subjects performed
facial immersion under the LBNP, LBPP, and control condi-
tions in random order. Arnold et al. (4) had their subjects
perform multiple vagal maneuvers on the same day and
allowed a minimum 3-min recovery interval between maneu-
vers. Thus we believe that performing multiple immersions
with the recovery interval described below caused possible
residual effects between conditions to be avoided. To avoid
the residual effects of exercise, the exercise trial was per-
formed on a separate day. The order in which sessions 1 and
2 were carried out was also randomized. All trials were
performed at an ambient temperature of 26°C. A schematic
representation of the experimental timeline is presented in
Fig. 1.

Session 1. Each condition began with a 5-min upright rest
period during which baseline readings were taken (precondi-
tion baseline resting). The baseline resting period was car-
ried out while the subject was in the pressure chamber to

Figure 1. Overview of protocol timeline for experimental session 1 [control, lower body negative pressure (LBNP), lower body positive pressure (LBPP) immersion conditions] and for experimental session 2 [postexercise hypotension (PEH) immersion]. $\dot{V}O_2\text{max}$, maximal oxygen consumption.
minimize the effect of posture across and within subjects for all measures. Subjects were then exposed to either −20 mmHg LBNP, +40 mmHg LBPP, or no lower body pressure (control). Once the subject was positioned within the pressure chamber and all cardiovascular parameters had stabilized, a 30-s baseline reading was recorded. Stabilization occurred in <5 min for all subjects. After baseline values had been registered under each condition, subjects then performed a 30-s apneic facial immersion in cold water (6.0 ± 1.2°C). The immersion time was predetermined and has been used by other studies (3, 5, 6). The water-filled container was placed below the subject’s chin such that total facial immersion including the forehead and chin was achieved by simple flexion of the neck. Facial immersions were performed during a midinspiratory breath hold, and subjects were cautioned to avoid the Valsalva maneuver (5, 6). Recordings were continued for 2 min postimmersion, and there was a 15-min interval between the end of facial immersion and the commencement of the 5-min baseline period of the next condition. Thus there was a 20-min interval between dives.

Session 2. Subjects reported to the laboratory and were fitted with an esophageal thermocouple and the skin heat flow sensors before a 5-min baseline recording of Tsk, Ttt, HR, SkBF, and MAP was performed. The subjects then performed 15 min of exercise on the treadmill at a work rate equivalent to 75% of their predetermined VO2 max. This intensity has previously been reported to be effective in eliciting PEH (23, 25). After exercise, the subjects were seated in the upright position until Tsk returned to preexercise values and a plateau in elevated Tsk was observed. That is, facial immersion was initiated when Tsk had returned to preexercise values, and when Tsk and cardiovascular measures were stable. The stabilization of Tsk, Tes, and cardiovascular measures occurred within 15–20 min postexercise and was consistent with the results of Kenny and Neidre (23).

Data Analysis

A 30-s resting average was calculated before any of the conditions were administered. The 30-s period between 60 and 30 s before facial immersion was used to calculate average baseline values of MAP, SkBF, and HR. An average relative value of the last 10 s of facial immersion was calculated for MAP and SkBF for comparison of total effect between conditions. However, an average relative value for each 5-s period during facial immersion was also calculated to show the time course of the response under each condition (3). The relative change from baseline was calculated for MAP and SkBF. For HR, however, only absolute values were also calculated for each 5-s period during facial immersion as well as an average value during the last 10 s of immersion. All values represent means ± SE for six subjects. A paired Student’s t-test was used for statistical analysis with differences being considered significant at P < 0.05.

RESULTS

The HR reached during the last 10 s of immersion was significantly different from baseline under all conditions (P < 0.05; Figs. 2 and 3). The average HR nadir measured during the last 10 s of immersion for LBNP (61 ± 1 beats/min) and PEH (80 ± 2 beats/min) was different from both LBPP (46 ± 2 beats/min) and control (47 ± 1 beats/min) (P < 0.05; Table 2). The onset of bradycardia occurred at different times during immersion. Control HR became significantly different from baseline at 10 s followed by PEH and LBNP at 15 s and then LBPP at 20 s (Fig. 3).

Each of the three conditions applied caused significant changes in HR from baseline resting before immersion (P < 0.05; Table 2). Thus the application of LBPP caused a 15 ± 1 beats/min decrease in HR, whereas LBNP and PEH caused a 9 ± 1 and a 13 ± 2 beats/min increase, respectively. Exercise also caused a 0.54 ± 0.11°C (P < 0.05) elevation in Tes from preexercise values that remained elevated during the time in which immersion was performed. MAP increased ~45–50% from baseline during immersion for all conditions (P < 0.05; Fig. 2A; Table 1). Exercise was effective in inducing PEH with a MAP decrease of 4.9 ± 0.6 mmHg compared with preexercise resting baseline (P < 0.05).

A marked reduction in SkBF was observed for all immersions at both the acral and nonacral sites (P < 0.05; Fig. 2, B and C; Table 1); however, there were no differences among conditions. The magnitude of the
reduction in flow was much greater at the acral than the nonacral site with reductions of −72 and −33%, respectively, for all conditions (P < 0.05; Fig. 2, B and C; Table 1).

**DISCUSSION**

This study was unique in examining the diving response during altered cardiac filling subsequent to modification by lower body pressure and exercise. The most important finding was that various conditions that reduce cardiac filling do attenuate diving bradycardia. The diving response was produced in each of the four conditions applied in this study; however, the pattern and magnitude of the responses differed significantly. These results support previous postulates that diving bradycardia may be attenuated due to reduced cardiac filling either as a result of exogenous heat stress (5) or as a result of high intrathoracic pressure (1).

During LBNP (7, 9) and PEH (29), efferent sympathetic activity is increased through a baroreflex arc to maintain peripheral vascular resistance, whereas during LBPP (13), cardiac parasympathetic activity is increased. Both apnea and cold stimulation by the water promote chronotropic parasympathetic stimulation via the vagus nerve (2). Thus performing apneic facial immersion during LBNP or PEH sets up two independent stimuli that give rise to conflicting inputs of a common effector at the level of the brain stem. Our results suggest that when sympathetic efferent activity to the heart is increased because of reduced cardiac filling, diving bradycardia is attenuated. As a result of increased efferent sympathetic activity, the diving induced parasympathetic response is either inhibited or attenuated, thus reducing the bradycardia observed. In addition to the previously mentioned influences on the diving response, it is important to note that feedback as a result of respiratory muscle inhibition and chemoreceptor stimulation is a key component of the response (14). It was assumed that such influences were not altered by the experimental variables used in our protocol. Feedback subsequent to respiratory muscle inhibition and chemoreceptor stimulation was considered to be standardized in our study with each dive being limited to 30 s.

Facial immersion during PEH resulted in the greatest attenuation in diving bradycardia. During PEH, not only is preload compromised as a result of blood pooling in the previously active musculature (15, 23, 24) but also systemic hypotension exists (16, 17). It is reasonable to assume that the magnitude of the cardiac efferent sympathetic activity generated during systemic hypotension (29) would be greater than that generated by cardiopulmonary baroreceptor unloading with no change in MAP. It is possible that because of this greater magnitude of cardiac sympathetic outflow during PEH that diving bradycardia showed a greater attenuation than with LBNP.

Although reducing cardiac filling resulted in an attenuation of bradycardia, increasing cardiac filling with LBPP did not amplify bradycardia beyond that of the control condition. In order for the heart to maintain cardiac output at low HR values, stroke volume must be increased. It is known that during diving bradycardia cardiac output falls because stroke volume cannot compensate totally for the reduction in HR. In the resting seated position with LBPP of +40 mmHg used in this study, stroke volume is maximized (28); therefore, regardless of increased cardiac filling, the magnitude of bradycardia may have been limited to maintain

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**Table 1. Mean percent change from predive baseline to mean value measured during last 10 s of immersion**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>LBNP</th>
<th>LBPP</th>
<th>PEH</th>
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</thead>
<tbody>
<tr>
<td>MAP</td>
<td>+47 ± 3</td>
<td>+47 ± 3</td>
<td>+47 ± 2</td>
<td>+48 ± 2</td>
</tr>
<tr>
<td>SkBF</td>
<td>†0.05</td>
<td>†0.05</td>
<td>†0.05</td>
<td>†0.05</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
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<th>LBPP</th>
<th>PEH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acral</td>
<td>†77 ± 3</td>
<td>†71 ± 5</td>
<td>†69 ± 8</td>
<td>†74 ± 3</td>
</tr>
<tr>
<td>Nonacral</td>
<td>†33 ± 3†</td>
<td>†32 ± 4†</td>
<td>†32 ± 2†</td>
<td>†34 ± 3†</td>
</tr>
</tbody>
</table>

Values are means ± SE given in % for 6 subjects. LBNP, lower body negative pressure; LBPP, lower body positive pressure; PEH, postexercise hypotension; MAP, mean arterial pressure; SkBF, skin blood flow. *Significant difference from acral skin blood flow reduction, P < 0.05.

**Table 2. Absolute heart rate values**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>LBNP</th>
<th>LBPP</th>
<th>PEH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Precondition</td>
<td>78 ± 1</td>
<td>77 ± 1</td>
<td>77 ± 1</td>
<td>78 ± 2</td>
</tr>
<tr>
<td>Baseline</td>
<td>86 ± 1*</td>
<td>62 ± 1*</td>
<td>91 ± 2*</td>
<td></td>
</tr>
<tr>
<td>Last 10 s</td>
<td>47 ± 1†</td>
<td>61 ± 1†</td>
<td>46 ± 2†</td>
<td>80 ± 2†</td>
</tr>
</tbody>
</table>

Values are means ± SE given in beats/min for 6 subjects. Baseline measurements represent the effect of the experimental condition on heart rate before immersion. Mean heart rates during the last 10 s of immersion are also shown. *Significant difference from resting precondition heart rate, P < 0.05. †Significant difference from baseline, P < 0.05.
an adequate cardiac output. The use of LBPP in this study was thought to attenuate cardiac sympathetic activity; however, it is important to consider the possibility that in the population studied tonic sympathetic activity was low or negligible and thus LBPP had no effect on diving bradycardia.

It should be noted that our control immersion produced the greatest effect on HR and that this effect was consistent with other studies using the same immersion time and similar water temperature (5, 6). At the other extreme, however, PEH exhibited the smallest bradycardic effect. However, the HR effects under the LBPP and LBNP conditions were also very different (Table 2). The average HR recorded in the last 10-s of LBPP and LBNP were also very different; that is, PEH > LBPP > control = LBPP.

In this study, we also investigated the differences in cutaneous blood flow in acral (hand) and nonacral (forearm) regions during cutaneous vasoconstriction. In acral regions, cutaneous arterioles are innervated only through noradrenergic sympathetic nerves, and thus reflex changes, whether thermoregulatory or non-thermoregulatory, are mediated by adjustments in vasoconstrictor tone (21). Nonacral skin, however, includes both a noradrenergic vasoconstrictor system (both \(\alpha_1\) and \(\alpha_2\)-receptors) and a separate active vasodilator system still under investigation (11, 12, 32). Whereas nonacral skin possesses two sympathetic neural pathways, the active vasodilator system is thought to play a prominent role during heat stress (21). To our knowledge, the potential differences between these two regions of skin have not been examined during facial immersion. Our data showed a greater percentage reduction in acral blood flow (~72%) than nonacral (~33%) when comparing either absolute values or when values were normalized to maximal cutaneous vascular conductance (data not shown). The results show that peripheral vasoconstriction was produced in both skin regions, but the reason for the greater percent reduction in acral skin is not completely clear. Further investigation is required to examine the differences between acral and nonacral regions. Specifically, it should be examined under conditions where blood flow levels during predive baseline are nearly equal between regions.

When examining the results globally, the fact that the experimental conditions resulted in an altered HR response but did not affect reductions in SkBF or the rise in MAP is noteworthy. Arnold and Nadel (5, 6) suggested that peripheral vasoconstriction is a requirement for diving bradycardia and noted that there is a relatively linear relationship between HR changes and changes to forearm blood flow. However, Finley et al. (10) previously concluded that the pharmologic blockade of vasoconstriction and the increase in MAP did not affect the reflex diving bradycardia. Thus the peripheral vasoconstriction and rise in MAP are not essential for diving bradycardia to occur but are merely coincidental (10). Our results show that despite the varying degrees of bradycardia as a result of the experimental conditions, the acute reduction in SkBF and rise in MAP were unaffected. This observation supports the idea that although both parasympathetic and sympathetic efferents are activated during facial immersion, the responses they effect are independent of each other.

The results show that reducing cardiac filling pressure and, to a greater extent, inducing mild hypotension through exercise will attenuate the cardiac parasympathetic effects normally exhibited during facial immersion. The fact that peripheral blood flow and MAP changes were not different between conditions supports the idea that, during facial immersion, cardiac and peripheral responses are exerted separately.

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REFERENCES


